

## WEST Search History





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		<i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR</i>	
<input type="checkbox"/>	L1	blood near group near binding	15
<input type="checkbox"/>	L2	hops or hop-s or hops	41251
<input type="checkbox"/>	L3	L2 and (helicobacter or pylori)	70
<input type="checkbox"/>	L4	L2 same (helicobacter or pylori)	15
<input type="checkbox"/>	L5	outer near membrane near (polypeptide or peptide or protein)	3608
<input type="checkbox"/>	L6	L5 and (pylori or pyloridis or pylon or pylorum or pyloris or helicobacter or heliobacter)	534
<input type="checkbox"/>	L7	L5.clm. and (pylori or pyloridis or pylon or pylorum or pyloris or helicobacter or heliobacter).clm.	11
<input type="checkbox"/>	L8	L7 not l1	11
<input type="checkbox"/>	L9	L8 not l4	10
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<input type="checkbox"/>	L12	L10.clm. and (helicobacter or pylori or pyroli or pyloridis or pylorid or pyloridis or hpylori)	5
<input type="checkbox"/>	L13	73.clm. or 74.clm. or 75.clm. or 76.clm.	100680
<input type="checkbox"/>	L14	L13 same (heliobacter or helicobacter or pylori)	31

END OF SEARCH HISTORY

DOCUMENT-IDENTIFIER: US 20050009037 A1

TITLE: *Helicobacter bizzozeronii* outer membrane protein encoding gene and its use in diagnostic and treatment methods

Summary of Invention Paragraph:

[0007] Several families of outer-membrane protein (Omp) have been identified in *H. pylori* (Alm et al., "Comparative Genomics of *Helicobacter pylori*: Analysis of the Outer Membrane Protein Families," Infect. Immun. 68:4155-4168 (2000)). One major family (*Hop*) contains thirty-two homologous members, some of which are functional as porins (Peck et al., "Characterization of Four Members of a Multigene Family Encoding Outer Membrane Proteins of *Helicobacter pylori* and Their Potential for Vaccination," Microbes Infect. 3:171-179 (2001)). Some outer-membrane proteins not in paralogous families have been also identified. Among them are small-molecular-mass bacterial peptidoglycan-associated lipoproteins (PAL) such as Lpp20 and Omp22 (Kostrzynska et al., "Molecular Characterization of a Conserved 20-Kilodalton Membrane-Associated Lipoprotein Antigen of *Helicobacter pylori*," J. Bacteriol. 176:5938-5948 (1994); Kim et al., "Cloning and Characterization of a 22 kDa Outer-Membrane Protein (Omp22) from *Helicobacter pylori*," Mol. Cells 10:633-641 (2000)).

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		<i>DB=USPT; PLUR=YES; OP=OR</i>	
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<input type="checkbox"/>	L6	l5 and (man or human or sapian or sapien)	67
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## CLUSTAL W (1.81) multiple sequence alignment

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### Graphical overview of the alignments

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Pfam hits 

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□ 1: FEMS Immunol Med Microbiol. 1994 Jun;9(1):15-21.

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### **Detection of microbial surface antigens that bind Lewis(a) antigen.**

**Essery SD, Weir DM, James VS, Blackwell CC, Saadi AT, Busuttil A, Tzanakaki G.**

Department of Medical Microbiology, University Medical School, University of Edinburgh, UK.

There is evidence that the Lewis(a) blood group antigen is one of the receptors for a number of potentially pathogenic microorganisms. To determine how widely distributed the microbial adhesins are that bind this antigen, anti-idiotypic antibodies produced against monoclonal anti-Lewis(a) were used in coagglutination assays to screen a variety of species. The following were agglutinated: 7/7 strains of *Staphylococcus aureus*; 10/19 (53%) strains of *Neisseria meningitidis*; 8/13 (62%) strains of *Haemophilus influenzae*; 1/3 strains of *Helicobacter pylori*; 1/2 strains of *Neisseria gonorrhoeae*; 1/2 strains of *Candida albicans*. The application of the anti-idiotypic antibodies to studies of host cell receptors, isolation of adhesins and development of new epidemiological typing reagents is discussed.

PMID: 7920460 [PubMed - indexed for MEDLINE]

A handwritten signature, 'Adonis', is enclosed within a hand-drawn oval. The signature is written in a cursive, flowing style.

Gastroenterology. 1997 Apr;112(4):1179-87.

Related Articles, Links

Gastroenterology

**Isolation of a cell surface component of *Helicobacter pylori* that binds H type 2, Lewis(a), and Lewis(b) antigens.**

**Alkout AM, Blackwell CC, Weir DM, Poxton IR, Elton RA, Luman W, Palmer K.**

Department of Medical Microbiology, University of Edinburgh, Scotland.

**BACKGROUND & AIMS:** Individuals of blood group O and nonsecretors of ABO blood group antigens are more susceptible to peptic ulcers. The aim of this study was to determine if blood group antigens associated with group O or secretor status are epithelial cell receptors for *Helicobacter pylori*. **METHODS:** Bacterial binding and binding of monoclonal antibodies to H type 2, Lewis(a), and Lewis(b) to Kato III, buccal epithelial, and gastric mucosal cells were shown by flow cytometry. Bacterial outer membrane proteins eluted from H type 2, Lewis(a), or Lewis(b) were shown by polyacrylamide gel electrophoresis. **RESULTS:** Kato III and human epithelial cells bound each monoclonal antibody; O cells bound more anti-H type 2 ( $P < 0.05$ ). Binding indices for *H. pylori* correlated with those for anti-H type 2 ( $P < 0.005$ ) and anti-Lewis(b) ( $P < 0.001$ ) but not anti-Lewis(a). A 61-kilodalton protein was eluted from H type 2, Lewis(a), or Lewis(b). **CONCLUSIONS:** Our results indicate that H type 2 is an important receptor for the 61-kilodalton bacterial adhesin, partly explaining increased susceptibility of individuals of blood group O to ulcers. Lewis(b) binds *H. pylori* more efficiently than Lewis(a). If these interactions occur in vivo, lack of Lewis(b) in mucosal fluids of nonsecretors may contribute to colonization by *H. pylori*.

PMID: 9098001 [PubMed - indexed for MEDLINE]

*Request*

□ 1: Gastroenterology. 1997 Apr;112(4):1179-87.

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**Gastroenterology**

**Isolation of a cell surface component of *Helicobacter pylori* that binds H type 2, Lewis(a), and Lewis(b) antigens.**

**Alkout AM, Blackwell CC, Weir DM, Poxton IR, Elton RA, Luman W, Palmer K.**

Department of Medical Microbiology, University of Edinburgh, Scotland.

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PMID: 9098001 [PubMed - indexed for MEDLINE]



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1: FEMS Immunol Med Microbiol. 1996 Dec 1;16(2):141-55.

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**ELSEVIER**  
**FULL-TEXT ARTICLE**

## Cell envelope characteristics of *Helicobacter pylori*: their role in adherence to mucosal surfaces and virulence.

Clyne M, Drumm B.

Department of Paediatrics, University College Dublin, Our Ladys Hospital for Sick Children, Crumlin, Ireland.

*Helicobacter pylori* colonises the gastric mucosa of humans and causes both antral gastritis and duodenal ulcer disease. Exactly how *H. pylori* causes disease is not known but several pathogenic determinants have been proposed for the organism. These include adhesins, cytotoxins and a range of different enzymes including urease, catalase and superoxide dismutase. Surface molecules of *H. pylori* such as flagella, lipopolysaccharide, the urease enzyme and outer membrane proteins are putative adhesin molecules. While phosphatidylethanolamine and the Lewis(b) blood group antigen have been proposed as receptor molecules for the organism the exact mechanism by which *H. pylori* adheres to the gastric mucosa has still to be identified. Characterisation of the adhesins of *H. pylori* could lead to the development of adhesin analogues for use in the inhibition of colonisation and improved therapy for ulcer disease. In vivo studies with isogenic mutants which are incapable of adhering to the gastric mucosa would greatly clarify the significance of adherence. Such mutants could possibly be useful as a vaccine against infection with wild-type organisms.

PMID: 8988394 [PubMed - indexed for MEDLINE]

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Feb 13 2006 12:53:38

□ 1: APMIS. 1992 Oct;100(10):908-13.

[Related Articles](#), [Links](#)

**Haemagglutination patterns of *Helicobacter pylori*. Frequency of sialic acid-specific and non-sialic acid-specific haemagglutinins.**

**Lelwala-Guruge J, Ljungh A, Wadstrom T.**

Department of Medical Microbiology, University of Lund, Sweden.

Thirty-two *Helicobacter pylori* strains were screened for haemagglutination (HA) activity with erythrocytes of 11 different animal species. Twenty-three strains (72%) that agglutinated human erythrocytes exhibited a broad-spectrum HA profile. Human, guinea pig and bovine erythrocytes high in sialoglycoconjugates were strongly agglutinated by most strains. Except for two, seven strains (22%) that did not agglutinate human erythrocytes exhibited a narrow-spectrum HA profile, commonly not inhibitable by sialoglycoconjugates or N-acetylneuraminlactose (NANLac). Strains were classified into three major HA classes. HA of 10 strains (31%) in class I was inhibited by different combinations of NANLac, orosomucoid or fetuin, but not by asialofetuin, suggesting the presence of sialic acid-specific HAs probably recognizing NeuAc alpha-(2-3)-Gal isomer. Twelve strains (38%) in class II exhibited a different receptor specificity binding to different combinations of NANLac, orosomucoid and fetuin, as well as asialofetuin. No inhibition was observed with 10 strains (31%) in class III; thus, this receptor seems different from both the other classes. Of 21 strains (66%) in classes I and II, HA of 11 strains (34%) was inhibited with NANLac, 14 strains (44%) with orosomucoid and 15 strains (47%) with fetuin. The great heterogeneity observed in HA patterns indicates that the HAs of different strains may recognize a heterogeneous class of sialoglycoconjugates on the erythrocyte membrane.

PMID: 1445697 [PubMed - indexed for MEDLINE]

□ 1: J Infect. 1992 May;24(3):263-7.

[Related Articles, Links](#)

### **Adherence of *Helicobacter pylori* to HEp-2 cells.**

**Figueroa G, Portell DP, Soto V, Troncoso M.**

Microbiology Unit, University of Chile, Santiago.

The adherence of 25 strains of *Helicobacter pylori* was evaluated in HEp-2 cells. These bacterial isolates, obtained from Chilean patients with gastric disorders, were also tested for haemagglutination of human red blood cells. Adherence of HEp-2 cells was expressed as a common property of all strains, irrespective of whether the cultures were grown on semi-solid or in liquid media. Previous reports that haemagglutinating activity was present in cells grown only on semi-solid media were confirmed. Adherence to HEp-2 cells was suppressed when bacterial cells were pretreated with homologous or heterologous whole human serum, containing specific antibodies of *H. pylori*. Adherence remained unaltered when bacterial cells were similarly treated with normal serum lacking specific antibodies. These observations imply that adhesions are expressed *in vivo* and suggest that an adherence mechanism, not depending on the expression of specific haemagglutinin antigen, operates for *H. pylori*.

PMID: 1602147 [PubMed - indexed for MEDLINE]

□ 1: FEMS Immunol Med Microbiol. 1995 Feb;10(3-4):307-16.

Related Articles, Links

**Attachment, ingestion and intracellular killing of *Helicobacter pylori* by human peripheral blood mononuclear leukocytes and mouse peritoneal inflammatory macrophages.**

**Chmiela M, Paziak-Domanska B, Wadstrom T.**

Department of Medical Microbiology, University of Lund, Sweden.

The different steps of phagocytosis, attachment, ingestion and intracellular killing of cells of *Helicobacter pylori* strain 17874 (expressing sialic acid-specific haemagglutinin) and cells of *H. pylori* strain 17875 (expressing non-sialic acid-specific haemagglutinin) have been studied. More cells of sialopositive *H. pylori* strain 17874 have been found attached to human peripheral blood mononuclear leukocytes (PBM) and mouse peritoneal inflammatory macrophages (PIM) than cells of sialonegative *H. pylori* strain 17875. Binding of cells of *H. pylori* strain 17874 has been significantly inhibited by treatment of phagocytes with neuraminidase. Inhibition of adhesion of these bacteria preincubated with foetuin to normal phagocytic cells has also been found. Well adhering cells of *H. pylori* strain 17874 were more resistant to killing mechanisms of human PBM and mouse PIM than cells of strain 17875. Good, probably sialic acid-specific haemagglutinin dependent, adhesion of *H. pylori* bacteria to phagocytes can be considered as an important virulence factor which facilitates the pathogen to avoid the defence mechanisms.

PMID: 7773248 [PubMed - indexed for MEDLINE]



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L1: Entry 11 of 15

File: USPT

Jun 25, 2002

US-PAT-NO: 6410719

DOCUMENT-IDENTIFIER: US 6410719 B1

TITLE: Blood group antigen binding protein and corresponding agents

DATE-ISSUED: June 25, 2002

## INVENTOR-INFORMATION:

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Ilver; Dag	Ume.ang.			SE

US-CL-CURRENT: 536/23.7; 536/23.1

## CLAIMS:

What is claimed is:

1. An isolated recombinant DNA from Helicobacter pylori encoding an adhesin protein, wherein said protein specifically binds to the fucosylated blood group antigens Lewis .sup.b and H-1, comprising the nucleotide acid sequence of SEQ ID NO:1.

2. A vector comprising an isolated recombinant DNA from Helicobacter pylori encoding an adhesin protein, wherein said protein specifically binds to the fucosylated blood group antigens Lewis.sup.b and H-1, comprising the nucleotide sequence of SEQ ID NO:1.

3. An isolated recombinant DNA from Helicobacter pylori encoding an adhesion protein, wherein said protein specifically binds to the fucosylated blood group antigens Lewis.sup.b and H-1, comprising the open reading frame of the nucleotide sequence of SEQ ID NO:1.

4. A vector comprising an isolated recombinant DNA from Helicobacter pyroli encoding an adhesin protein, wherein said protein specifically binds to the fucosylated blood group antigens Lewis.sup.b and H-1, comprising the open reading frame of the nucleotide sequence of SEQ ID NO:1.

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L1: Entry 9 of 15

File: USPT

Mar 23, 2004

US-PAT-NO: 6709656

DOCUMENT-IDENTIFIER: US 6709656 B1

TITLE: Helicobacter pylori adhesin binding group antigen

DATE-ISSUED: March 23, 2004

## INVENTOR-INFORMATION:

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Ilver; Dag	35 Umea			SE

US-CL-CURRENT: [424/190.1](#); [424/184.1](#), [424/234.1](#), [435/252.1](#), [435/7.32](#), [514/25](#),  
[530/350](#)

## CLAIMS:

What is claimed is:

1. An isolated and purified bacterial blood group antigen binding adhesin protein (BabA) from Helicobacter pylori species, wherein said protein binds specifically to fucosylated Lewis.sup.b type I and H-1 blood group antigen-glycoconjugates and, wherein said protein contains less than 20% bacterial protein impurities, has a molecular weight in the interval of 73 to 75 kDa as determined by sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE), and is not a HopA, HopB, HopC, HopD, or HopE protein.

2. The adhesion protein of claim 1, wherein the SEQ ID NO:5

EDDGFYTSVGYQIGEEAQMV

is in an amino terminal position.

3. The adhesion protein of claim 1, wherein the protein has a molecular weight of about 73.5 kDa, as determined by SDS-PAGE.

4. The adhesion protein of claim 3, wherein said Helicobacter pylori species is Helicobacter pylori strain CCUG 17875.

5. An immunogenic composition comprising an adhesion protein according to any one of claim 1, 2, 3, or 4, optionally together with pharmaceutically acceptable excipients.